

two weeks leukocytes declined to 30,000 and rose again to 72,000 toward the end of the period. A rapid decline eventuated in death on January 15, 1928. A total of fifty-two x-ray treatments in four series were given, the technical details and comments being omitted.

Autopsy six hours after death. Gross and histopathological diagnosis: Pulmonary edema, passive congestion and hypostatic pneumonia; gelatinous degeneration of subepicardial fat; chronic interstitial myocarditis, dilatation and chronic mural endocarditis; hemoperitoneum; massive splenomegaly of myeloid leukemia; passive congestion, portal sclerosis and leukemic infiltration of liver; chronic cholecystitis; congestion of gastro-intestinal tract; chronic interstitial pancreatitis; chronic nephrosis; lipoidal changes in adrenals; chronic bronchial, lumbar and mesenteric lymphadenitis; chronic interstitial thyroiditis; leukemic infiltration of rib marrow.

Spleen: weight, 10,110 grams (22¼ pounds); dimensions, 42 x 27 x 15 centimeters. Liver: weight, 1840 grams. An extensive review of literature has failed to reveal in leukemia a splenomegaly of these dimensions and weight.

Southern Pacific General Hospital.

POLYCYTHEMIA COINCIDENT WITH ACTIVE PULMONARY TUBERCULOSIS

REPORT OF CASE

By HENRY L. HOLZBERG, M. D.
San Francisco

A THOROUGH search of the available literature reveals no reported cases of polycythemia in human beings suffering from pulmonary tuberculosis. However, there have been reported a number of cases of polycythemia in which a tuberculous spleen was found. As we know, primary tuberculosis of the spleen is extremely rare, whereas secondary tuberculosis of the spleen is fairly common.

REVIEW OF THE LITERATURE

In 1899 Rendu and Widal¹ described a symptom complex, including tuberculosis of the spleen, polycythemia without leukemia and with cyanosis. They attributed this combination to the splenic condition. This case was of eight years' duration and showed no pulmonary or glandular involvement. The red cells numbered 6,200,000 and the white cells 6000. Cyanosis was present. At autopsy the spleen weighed 3780 grams. Evidences of tuberculosis were found in the spleen, liver, right lung, mesenteric glands, pancreas, and kidneys. This case is comparable to the case reported here.

In 1904 Bayer² reported a series of nine operative cases of polycythemia with seven recoveries, including one of his own, and nineteen autopsy cases. He stressed splenectomy in these cases. His patient recovered after splenectomy. It was of six weeks' duration. The red blood cell count was 6,000,000 and the white cell 7500. The hemoglobin was 40 per cent. In this patient there was no pulmonary or glandular involvement. There was no cyanosis.

In 1912 Winternitz³ summarized fifty-one cases in an article on tuberculosis of the spleen. The red blood count was given in twenty-six cases. It

was normal in nine (35 per cent); below normal in eleven (42 per cent); above normal in six (23 per cent). In acute generalized tuberculosis the spleen is usually enlarged. In pulmonary tuberculosis of adults it is rarely enlarged, and it is usually atrophied and diminished in volume. In these fifty-one cases, a note on the lungs was found in forty cases. Of these latter forty cases there were negative clinical findings in twenty (50 per cent); a quiescent lesion was found in three cases (7½ per cent); signs of pulmonary involvement developed in three cases (7½ per cent); two cases showed chronic pleurisy (5 per cent); and five cases showed extensive pulmonary involvement (12½ per cent). In the latter five cases three of them developed signs of pulmonary involvement apparently secondary to the spleen.

Polycythemia was first described as a definite disease by Vaquez in 1892. In 1903 Osler called general attention to this condition. Since that time many cases have been reported. Many etiologic suggestions have been made. Among these may be mentioned splenic tuberculosis and the theories of blood stasis, the extreme viscosity of the blood being a marked phenomenon in these cases of polycythemia.

In 1914 Douglas and Eisenbray⁴ reported a case of tuberculous spleen and polycythemia; autopsy showed small, discrete tubercles in the lungs.

Weiss⁵ in 1924 reported a case of polycythemia in which the spleen was not enlarged, but in which x-rays showed infiltrated lungs. No mention of tuberculosis was made.

Antonelli⁶ in 1927 reported a 62-year-old male who had polycythemia rubra and an incomplete Addisonian syndrome with an evident carcinomatous neoplasm of the upper lobe of the right lung, with metastases. No evidences of tuberculosis present.

REPORT OF CASE

R. S., age twenty-one, American-born Hebrew, first consulted me February 1922. His complaint at that time was hemorrhage.

His family history was negative except his first cousin on his father's side gave history of an occasional prolonged bleeding following very slight cuts, such as razor cuts, and epistaxis. There was no other family history of bleeders, hemophilia, polycythemia, tuberculosis, carcinoma, syphilis, jaundice, or insanity.

Past History.—Born in San Francisco. Occupation: Real estate salesman. Denied all diseases except varicella. No venereal history. He used no coffee or tea, alcohol, tobacco or drugs. There had been no previous operations.

Patient claimed he had always been well, strong and healthy. He was very active in football, and during a scrimmage his chest was apparently injured when fourteen men were piled on top of him.

Within twenty-four hours he suffered from a severe pulmonary hemorrhage. The doctor whom he consulted prescribed hot baths, but his hemorrhages continued. He went to a sanatorium, where he remained for six months. He was told he had pulmonary tuberculosis. Several sputum tests were made, all of which proved negative. No x-ray examinations were made.

After leaving the sanatorium he took unusually good care of himself and gained in weight. He noticed, however, whenever he sustained a slight cut that the

flow of blood was stopped only after considerable difficulty.

Upon blowing his nose during a coryza in February 1922, a severe hemorrhage ensued. His physician found diseased tonsils, and advised removal.

Several days later the writer was called in to see the patient. The hemorrhages had become more frequent and profuse. Patient claimed that he could feel a sensitive spot on the left side of his larynx whenever the blood trickle started and that within a few minutes a considerable hemorrhage, usually a pint or more of blood would be spat out without any effort. As a rule no clots were present.

Examination at that time showed a full-blooded young man raising small amounts of blood on any slight exertion, such as coughing, sneezing, etc. Temperature 101 toward evening, and a rapid, full pulse of 120. Respirations were not increased; no chills or pain; no history of vomiting, hematemesis, or tarry movements; tongue dry and coated; tonsils diseased, with the crypts full of cheesy matter. At that time there was no cyanosis. His chest showed active bilateral tuberculosis. X-ray examinations showed an old tuberculous condition at the left top. The blood count showed 96 per cent hemoglobin; 4,800,000 red cells with normal morphology; 10,800 white cells with 78 per cent polymorphonuclears. The coagulation time was fifteen minutes, whereas the normal is four to five minutes. The Wassermann and Noguchi control tests were negative. Urinalysis negative. No red blood cells found. Stools were negative for occult blood.

The hemorrhages were controlled within five days. Pituitrin intramuscularly and calcium chlorid intravenously were used. All symptoms cleared up. On February 3, 1922, patient was examined by Doctor Tobriner, who reported finding a deviated septum, adenoids, bad tonsils, and negative larynx. No varices, ulcers or bleeding points were seen. Fluoroscopy by Doctor Bryan on March 4, 1922, showed a question of old tuberculosis of the left top and no roentgen evidence of pathology in the gastro-intestinal tract.

Patient was given a series of calcium cacodylate injections intravenously. These brought his coagulation time down to normal. In November 1922 he brought up a small amount of blood while coughing. His coagulation was practically normal up to April 1923.

Patient was again seen on April 11, 1923. He contracted a slight cold, and during a coughing spell brought up several ounces of bright red blood. During the next three weeks a certain amount of oozing with an occasional clot occurred. The usual remedies were used, including calcium chlorid, pituitrin, thromboplastin (Squibb). On April 16, 1923, his blood coagulation time was normal, four minutes and fifteen seconds. Despite this, the bleeding continued off and on. There was no fever. Patient was moved to the Mount Zion Hospital in an ambulance.

While in the hospital the patient ran a slight fever toward evening. His blood count now showed 75 to 80 per cent hemoglobin; 5,000,000 red blood cells; the white count varied from 11,000 to 16,000; the polymorphonuclears, on entrance, were 82 per cent, and later dropped to 52 per cent. The coagulation time was four and one-half minutes. The urine was negative. X-ray graphs showed pulmonary tuberculosis evidenced by coarse mottling of both apices. Sputum examination revealed a few tubercle bacilli in two out of four samples. Patient was given normal horse serum intramuscularly. Slight amount of blood was brought up during the first five days. After that no further bleeding occurred, and the temperature became normal.

In the sputum examination the long type of acid-fast tubercle bacilli predominated in the positive specimens. His red blood cell count was over 5,000,000, despite considerable loss by hemorrhage. There was a beginning suggestion of cyanosis on the face, ears and fingers. A tendency toward clubbing was noticed.

Patient was kept under observation and got along very well for the rest of the year. His coagulation

time was around five minutes. His weight was 190 to 192. In January 1924 he had an attack of severe pain in his upper abdomen. There was a slight amount of fever and considerable vomiting of bile-stained matter. There were no hemorrhages.

In February 1924 the ears and throat were again examined by Dr. A. Houston. A ruptured right ear drum with diminished hearing, very badly infected tonsils, marked granular pharyngitis, and chronic rhinitis were evident. The larynx showed secondary catarrhal inflammation; the laryngeal vessels were large and engorged, and could easily rupture with a cough or sneeze. X-ray examination of the sinuses was negative.

From February 1924 up to July 1926 patient was in fairly good condition. He watched his temperature and there was no elevation except when he contracted a cold. Coughing would always be accompanied by small amounts of blood-stained mucus, but no actual hemorrhages occurred in this interval.

In July 1926 patient contracted a cold. One week later suddenly vomited more than a quart of blood. This was the first time he had ever vomited blood. During the first week he refused to come to the hospital, and in addition to the remedies used before, fibrogen and coagulen were used. There was very little improvement and the patient continued to vomit large quantities of blood. It is estimated he lost at least 3500 cc. during this week.

Physical examination at that time showed a well-developed and nourished young man. The cheeks were flushed and the skin slightly cyanosed, otherwise negative. Sclerae negative. Mucous membranes negative. Pupils equal; regular; circular; reacted normally to light and accommodation. Ophthalmoscopy revealed congested retinæ and engorged pulsating vessels over both disks. Eye muscles, ears, nose apparently negative. Lips cyanosed. Teeth apparently in good condition. Tongue clean, moist, protruded in median line without tremor. Tonsils badly infected, hypertrophied and congested. Pharynx congested. Superficial lymph glands negative. No supraclavicular or epitrochlears. Thyroid apparently negative.

Chest wall was negative; nipples likewise. Lungs showed roughened breath sounds with higher pitched and prolonged expiration found over both apices and both upper lobes, left more than right, posteriorly more than anteriorly. Similar findings in scattered areas throughout the rest of the lungs. Many moist râles. Vocal and tactile fremitus increased. Marked dullness around both lung roots. Both bases moved well and equally. No evidences of pleurisy or pleural effusion. Heart apex impulse not seen nor felt. Area of cardiac dullness within normal limits, extending to midclavicular line in fifth space; second rib above; right border under sternum. Sounds regular; good quality; A2 equals P2, neither abnormal; no murmurs. Pulse rate 90 to 110. Radials equal; regular; good volume; artery walls soft. Blood pressure: systolic, 90 to 108; diastolic, 60. Back negative. No limitation of motion. No kyphosis.

Abdomen smooth, level, soft, symmetrical, tympanitic throughout. No masses, tenderness or herniae. Not tender at either costovertebral angle. Liver dullness extended in midclavicular line from sixth rib to costal margin; edge not felt. Spleen and kidneys not felt. Inguinal glands negative.

Genitalia negative. Anus negative. Rectal examination negative.

Extremities negative, except for cyanosis of the finger nails with slight tendency to clubbing. No varices. No purpuric spots or petechiae.

Reflexes active. Knee, ankle, abdominal superior and inferior, cremasteric, brachial, biceps, triceps present and equally active on both sides. No ankle or patellar clonus. No Babinski, Gordon, Oppenheim, Chvostek or Trousseau. No Romberg.

The laboratory findings were as follows:

Catheterized urine negative; no red blood cells seen. Stool negative for occult blood. Coagulation time, two

minutes. Bleeding time, two and one-half minutes. Sputum was blood-stained and showed numerous acid-fast bacilli. Blood count showed 84 per cent hemoglobin; 5,600,000 red blood cells; 8500 white blood cells, with 71 per cent polymorphonuclears.

Five per cent glucose and 2 per cent soda was given by Murphy drip. This was in addition to the usual hypodermics. Morphine sulphate was freely used. Ice-bags were kept on the chest and abdomen.

On July 2, 1926, Dr. H. C. Moffitt saw the patient in consultation. He thought of a possible tuberculous spleen. He suggested removing the ice bags and removing the soda from the drip. He also suggested a platelet count and x-ray later.

During the next four days patient gradually improved and all vomiting of blood ceased. The laboratory findings were roughly the same. The platelet count varied from 225,000 on July 4, 1926 to 375,000 on August 3, 1926. Patient had a slight elevation of temperature from July 6, 1926 to August 1, 1926. No fever at any other time.

Fluoroscopy by Dr. L. Bryan on August 3, 1926, showed coarse mottling throughout both the left lung and at the right tip with beginning fibrosis; no evidence of organic lesion in the stomach.

During the next six weeks patient gradually improved. He was up and around and developed a cough with some bloody sputum at that time. This responded to a few 3.0 cc. injections of 10 per cent calcium chloride intravenously. His weight at that time was 173, and one month later 182.

Patient was again seen in April 1927. He was feeling well and weighed 200 pounds. Cyanosis was more marked on the cheeks and fingernails; the latter being definitely clubbed. Patient decided to go to Long Beach for a few months.

Was seen again September 15, 1927. Weight was 206. General feeling was good. Chest showed definite evidences of tuberculous involvement.

About the middle of October the patient had a slight cold and expectorated two clots of blood. He was not sure whether he brought this up or whether it came out of the back of his throat. However, there was no coughing, vomiting, or fever.

Ten days later he had a similar experience. He brought up about two ounces of bright frothy blood. Following this he began to have fever daily, reaching as high as 101 degrees either in the morning or evening. There were no chills or sweats, and no further hemorrhages. There was no coughing. Because of elevation of temperature with rapid pulse and high rate of respiration he was brought to the hospital by ambulance.

Positive findings at that time revealed a temperature up to 101, with pulse averaging 110 to 136, and respirations from 36 to 44. He was somewhat cyanosed. Tonsils were hypertrophied and congested. Tongue had a deep, dry, brown coat. Chest showed evidences of active tuberculosis, especially in the left upper lobe, with increased resonance, harsh breath sounds with prolonged high-pitched expiration and numerous râles. Signs were less marked on the left side. There was no evidence of fluid. Fingers were clubbed, and finger nails were markedly cyanosed. Abdomen was negative. Spleen was not palpable.

Course in Hospital.—Patient was very difficult to manage, refused coöperation, and did not like any of the treatment. He refused hypodermics and these had to be forced. At times he found it quite difficult to void urine and did not do so until threatened by catheterization.

Laboratory examination at this time showed hemoglobin 95 to 103, with red blood cells always over 5,000,000, with no leukocytosis. His bleeding time was three minutes; coagulation time, four minutes (apparently normal). Stools positive for occult blood. Sputum and blood clots were loaded with acid-fast bacilli. The urine was negative, except for a very slight trace of albumin. No blood cells.

After being in the hospital four or five days condition apparently became normal. Temperature was

normal and subnormal. Pulse dropped to 80; respirations to 20. He seemed to feel better. Chest condition seemed improved and patient was allowed to go up on the roof esplanade, and thought of returning to his home.

On November 11, 1927, patient awoke suddenly at 1 a. m. with a very sharp pain in the upper left half of his chest. A sudden gush of blood came up; at least a pint must have been brought up. Later on in the morning he had a chill, with temperature rising to 104 degrees. His pulse became very rapid and thin (146). Respirations were 45. From that time on patient ran an elevated temperature daily; it occasionally touched normal, but averaged 100 to 101 every day. At times his temperature seemed to be elevated in the morning and again in the evening. His pulse remained rapid, averaging 110. Respirations, around 40. Most of the time his blood pressure was systolic 100 to 110; diastolic 80, although at one reading it was 160 systolic and 80 diastolic. Patient continued to bring up small quantities of blood for a few days. Some relief apparently was obtained from calcium chloride intravenously and hemostatic serum by mouth. At times the patient became irrational, especially at night, and restraint would be necessary. He complained of severe headaches. He seemed to be troubled a great deal with a continual sensation of nausea which seemed to be from some irritation in the back of the throat. However, no treatment locally was of any benefit. Mercurochrome was applied to the tonsils.

Numerous consultations were had. On November 20, 1927, consultation with Doctor Jellinek, who recommended quiet, rest, observation, and x-ray. X-ray the following day showed extensive coarse mottling through both lung fields with suggestion of cavity behind the second left rib. On November 23, 1927, consultation had with Doctor Eloesser by the patient's request, and, although assured that there was nothing surgical in the case, he asked Doctor Eloesser to see him again two or three times. Consultation was had with Dr. Harold Hill and later with Doctor Rosenkrantz. Both commented on the unusual features of the case and presence of polycythemia.

Portable x-ray of chest and abdomen, taken on December 6, 1927, revealed an apparent miliary tuberculosis of both sides with no evidences of fluid. No evidence of enlarged spleen.

The condition quieted down, the patient felt fairly comfortable and appetite improved. However, the temperature remained up. Blood count showed a continued red blood cell count of over 5,000,000, despite the continued slight hemorrhages. The urine remained clear. Platelet count was 185,000. At times the patient developed pain in the left lower axilla suggestive of pleurisy, but no friction rub could be heard. Relief was obtained with local applications of mustard.

Patient became discouraged and insisted on going home. It was agreed to take him home from the hospital. However, against the writer's wishes, he was moved across the bay to Ross, by ambulance, on December 21, 1927.

He stood the trip apparently well. The condition remained about the same. On December 31, 1927, he suddenly became delirious. The temperature reaching 104.5 rectally. It was interesting that, during his delirium, he recited his football experiences. Patient died the next morning.

His last blood count, taken on December 22, 1927, showed 4,900,000 red cells with 90 per cent hemoglobin, and 12,100 white cells with 67 per cent polymorphonuclears.

It was extremely unfortunate that no autopsy could be obtained.

COMMENT

Briefly, what has been described was a condition of developing polycythemia in a patient with active, progressive, pulmonary tuberculosis. There

was no history of tuberculosis in the family. Active signs of tuberculosis developed suddenly following hemorrhages due to a crushed chest received in a football game. Despite treatment the pulmonary condition gradually progressed, and polycythemia appeared.

It is interesting to note that this patient followed instructions explicitly. He watched his temperature and weight. Dietetic orders were complied with and he gradually increased in weight. Despite this increase in weight and good care, x-ray pictures taken at varying intervals revealed a steady progress of his active pulmonary tuberculosis. The patient probably lived as long as he did because of the care he took of himself.

During the course of this disease he had several hemorrhages. On different occasions the blood apparently came from the throat, lungs, and stomach. No evidence of bleeding points were found in the nose, throat or larynx. Gastrointestinal fluoroscopy was negative.

The amount of blood lost in these hemorrhages was considerable. However, his red blood cell count averaged five to six million and more. The spleen was not palpable at any time, and apparently was not enlarged. There was no splenic shadow seen in the x-ray.

Very little is known about polycythemia. The old idea of overfeeding, increasing weight and adding resistance to aid the body help fight battles, such as this case of tuberculosis, seems not to have worked well in this particular case. It delayed the activity of the tuberculosis very little, if any; and in the meantime it encouraged whatever tendencies there may have been toward the development of the polycythemic condition.

It seems strange that there have not been more cases of this nature reported in the literature. It may be that blood counts have averaged well over five million following hemorrhages in tuberculous patients, in other plethoric individuals.

The therapy, naturally, was very difficult. In this patient, one was working between two directly opposite conditions. In tuberculosis it is advisable to build up the resistance and guard against loss of weight. To do this it is customary to recommend the best and richest of foods. In polycythemia we have an overthickening of the blood, and a rich blood-building diet would be contraindicated. So what improves one condition aggravates the other. Any roentgen therapy to benefit the polycythemic condition could not have been considered here. However, it was the tuberculous condition that was causing the acute symptoms, and the treatment was stressed in that direction.

It would be interesting to know how much of the tendency toward spontaneous (?) hemorrhage may be blamed on the polycythemic condition. His blood pressure was not elevated.

All these symptoms started after his chest injury. His previous history was uneventful. This may be considered as a case of latent pulmonary

tuberculosis which became activated through the injury.

In a personal communication Dr. H. C. Moffitt tells me that he is observing a woman with similar hemorrhagic tendencies and advanced tuberculosis in whom blood regenerates rapidly. He suggests calling the case reported herewith one of polycythemia rather than polycythemia vera.

SUMMARY

1. A latent tuberculous condition was activated by a football chest injury.
2. Despite excellent care activity gradually progressed.
3. Hemorrhages—apparently spontaneous—occurred from the nose, throat, lungs, and stomach at different intervals.
4. The red blood count was over 5,000,000, even after severe hemorrhage.

890 Geary Street.

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MASSIVE PULMONARY ATELECTASIS*

REPORT OF CASES

By MILTON J. GEYMAN, M. D.
Santa Barbara

MASSIVE pulmonary atelectasis is a well-known complication in a variety of conditions. Collapse of one or more lobes, with resulting displacement of the mediastinum, trachea, and heart, is the essential definition of this most interesting pulmonary complication. This must not be confused with the lung collapse associated with spontaneous or induced pneumothorax.

ETIOLOGY

A brief review of the known etiological factors might be of interest. In 1910 Pasteur described a series of massive atelectases occurring as a complication of diphtheria, and he believed them to be the result of postdiphtheritic paralysis. Chevalier Jackson¹ has shown this diphtheritic complication to be due really to bronchial obstruction by diphtheritic exudate.

Pasteur later reported sixteen cases of post-operative massive atelectases and stated that this comprised about 8 per cent of postoperative complications. Norris and Landis have described it as occurring with pneumonia.

The observation of wounded cases in military statistics has shown a high incidence of atelectasis of the contralateral type; that is, a collapse of one or more lobes when the trauma has occurred on the opposite side of the chest. The wound may have been not more than subcutaneous and amaz-

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